**In vivo** Insulin sensitivity and lipoprotein particle size and concentration in black and white children.

Short title: Lipoproteins and insulin sensitivity in youth

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Additional information for this article can be found in an online appendix at [http://care.diabetesjournals.org](http://care.diabetesjournals.org)

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**Objective:** To examine gender specific black/white differences in lipoprotein profile and the role of visceral adiposity, and to assess the relationship between insulin sensitivity and lipoprotein profiles in each group.

**Research Design and Methods:** Fasting lipoprotein particle size and concentration and visceral adipose tissue (VAT) were determined in 226 children (117 black, 101 male) aged 8-<18 years. The relationship between lipoproteins and insulin sensitivity was evaluated in a subset of 194 children (100 black, 88 male) who underwent a hyperinsulinemic-euglycemic clamp.

**Results:** Black males had smaller very low density lipoprotein (VLDL) and black females had larger high density lipoprotein (HDL) size than their white counterparts. Overall, blacks had larger low density lipoprotein (LDL) with no gender-specific race differences. After adjusting for VAT and gender, only VLDL size and concentrations remained significantly favorable in blacks. Analysis of lipoprotein particle size and concentration across insulin sensitivity quartiles revealed that in both racial groups, the most insulin resistant children had higher concentrations of small dense LDL, small HDL, large VLDL, and smaller LDL and HDL sizes than their more insulin sensitive counterparts.

**Conclusions:** The previously reported favorable lipoprotein profiles in black vs white children is partly due to race differences in VAT. In both groups however, the most insulin resistant youths have a high risk atherogenic profile of small dense LDL, small HDL and large VLDL, akin to the atherogenic lipoprotein pattern in adults with coronary artery disease.
**Type 2 diabetes mellitus (T2DM) and insulin resistance in children are associated with dyslipidemia (1,2), characterized by elevated triglycerides (TG) and low density lipoprotein (LDL) cholesterol, and low concentrations of high density lipoprotein (HDL) cholesterol (1-3). In addition to traditional lipid profiles, evidence suggests that insulin resistance and T2DM are associated with changes in lipoprotein particle size and subclass concentration (2,4). This is important to assess as traditional lipid measurements only partially predict disease risk (5). Recently, the SEARCH for diabetes in youth study (2) reported 36% of youth with T2DM and 62% of the poorly controlled ones had small dense LDL. Similarly, low proportions of large and high proportions of small HDL particles are found in children with T2DM and overweight, insulin resistant children (4). However, while some investigators report associations between LDL (6,7), HDL (8) and VLDL (6) particle size and fasting insulin, others do not (9). The high TG and low HDL cholesterol together with small, dense LDL in children with T2DM and insulin resistance is similar to the atherogenic lipoprotein phenotype in adults with coronary artery disease (10,11).

Black children despite being insulin resistant and hyperinsulinemic (12,13) compared with their white counterparts, have favorable lipid concentrations including lower LDL and TG, higher HDL (3,14,15), larger HDL and LDL, and smaller VLDL particles, and favorable lipoprotein subclass concentrations (6,8). Why black children have favorable lipoprotein profiles despite insulin resistance is not clear. One explanation could be lower visceral adiposity in black than white children despite similar overall adiposity (15). In black adults insulin resistance is not a good marker of TG or HDL-cholesterol concentrations, or lipoprotein particle size (16). Thus, the relationship between in vivo insulin sensitivity and lipoprotein profiles in black and white children needs to be examined if at risk children are to be identified for early treatments to improve lipoprotein profiles, and if those treatments are to be pertinent in children of different ethnicity. The present study, therefore, determined lipoprotein particle size and subclass concentrations in black and white children and measured in vivo insulin sensitivity to test the following hypotheses: 1) the favorable lipoprotein phenotype in black children is likely due to lower VAT compared with whites, and 2) the relationship between insulin sensitivity and lipoprotein profile is similar between black and white children.

**METHODS**

**Subjects:** Participants, 8-<18 years old, consisted of 117 black and 109 white normal-weight and overweight otherwise healthy children, except for 16 girls (10 white, 6 black; all BMI ≥98th percentile) with untreated polycystic ovary syndrome (PCOS). Some participants were reported before as part of an ongoing NIH-funded RO1 grant investigating race-related differences in childhood insulin sensitivity and secretion (3,12). Studies took place at Children’s Hospital of Pittsburgh NIH-funded Pediatric Clinical and Translational Research Center after IRB approval. Participants and their parents gave written informed consent. Of the 226 youth 194 had a hyperinsulinemic-euglycemic clamp. Exclusion criteria included diagnosed diabetes and use of medications that influence glucose, lipid metabolism or blood pressure. Participants' health was assessed by medical history, physical examination and hematological and biochemical tests. Pubertal development was assessed using Tanner criteria.

**Methods and Procedures:** Body weight and height were measured using
standardized equipment. Waist circumference was obtained at the midpoint between the lowest rib and the iliac crest (17). Body composition and abdominal adiposity were assessed by DEXA and computed tomography respectively, as described previously (12). Fasting blood samples were collected on all 226 children for analysis of lipoprotein particle size and concentration.

**In Vivo Insulin Sensitivity:** A subset of children (100 black, 94 white; including 16 girls with PCOS) underwent a 3-hr hyperinsulinemic-euglycemic clamp after 10-12 h of overnight fasting. Briefly, intravenous crystalline insulin (Humulin; Lilly, IN) was infused at a constant rate of 40 mU/m$^2$/min in normal-weight, and 80 mU/m$^2$/min in obese subjects, to suppress hepatic glucose production, as previously described (12). Plasma glucose was clamped at 5.6 mmol/L with a variable rate infusion of 20% dextrose based on arterialized plasma glucose determined every 5 min.

**Biochemical Measurements:** Plasma glucose was measured using a glucose analyzer (Yellow Springs Instruments, Yellow Springs, OH), and insulin concentrations by radioimmunoassay (12). Plasma lipid concentrations were determined using the standards of the Centers for Disease Control and Prevention as described previously (18), and lipoprotein particle size and subclass concentration using nuclear magnetic resonance spectroscopy (LipoScience Inc., Raleigh, NC) (19).

**Calculations:** Insulin-stimulated glucose disposal was calculated using the average exogenous glucose infusion rate during the final 30 min of the clamp (12). Insulin sensitivity was calculated by dividing insulin-stimulated glucose disposal rate by steady-state plasma insulin concentrations during the last 30 min of the clamp, as previously described (3,12).

**Statistical Analysis:** Independent t-tests, or Chi square for categorical variables, were used to examine race-related differences in subject characteristics and lipoprotein particle size and concentration in the group as a whole and stratified by gender. ANCOVA was used to determine the influence of gender and visceral adiposity on race-related differences in lipoprotein particle size and concentration. Black and white subjects were divided into insulin sensitivity quartiles by gender. One-way ANOVA or the nonparametric Kruskall-Wallis test, based on the nonviolation of statistical assumptions, was used to compare differences in lipoprotein particle size and concentration among quartiles. Tukey’s post-hoc comparison was used to identify differences among quartiles. As insulin sensitivity changes with puberty we analyzed differences among quartiles with an ANCOVA including Tanner stage as a covariate. Data were also analyzed excluding the 16 girls with PCOS to determine whether this condition may have affected our results. Stepwise multiple regression was used to assess the contribution of race, gender, age, insulin sensitivity and VAT to lipoprotein particle size. Data are presented as mean ± SEM with significance at P<0.05.

**RESULTS**

**Gender Specific Race-Related Differences:** Participant characteristics are summarized in Table 1. Black and white youth had similar body weight, BMI, body composition, and subcutaneous abdominal adipose tissue. VAT was lower in black than white girls with a similar tendency in black vs white boys (P=0.055).

As a group, black children had lower VLDL, total LDL and small dense LDL, higher large HDL concentrations; and larger HDL and LDL and smaller VLDL particle sizes than whites (see Table A1 in the online appendix available at http://care.diabetesjournals.org). After correcting for gender and VAT, race-
differences remained in VLDL particle size and total, large and medium VLDL concentrations, and total HDL concentrations (Online Appendix Table A1).

Table 2 depicts race data grouped by gender. Black males had smaller VLDL particle size, and black females had larger HDL size compared with their white counterparts. After adjusting for VAT differences in VLDL particle size remained in males \((P=0.028)\) and for HDL particle size persisted in females \((P=0.084)\). Black males and females had lower concentrations of total, large and medium VLDL, and black males had lower concentrations of total, small and very small LDL vs their white counterparts. After adjusting for VAT differences in VLDL remained in both genders but differences in LDL disappeared in males.

**Insulin Sensitivity, Lipoprotein Particle Size and Concentrations:** Figure 1 depicts lipoprotein particle size by *in vivo* insulin sensitivity quartiles. Irrespective of gender, black and white children in the lowest quartile of insulin sensitivity had smaller HDL (Figure 1A) and LDL (Figure 1B) size than children in the upper quartiles. White males in the lowest two quartiles of insulin sensitivity had larger VLDL particle size (Figure 1C) than their counterparts in the upper quartiles.

Figures 2-3 depict lipoprotein particle concentrations by *in vivo* insulin sensitivity quartiles. In white males and both genders for blacks, children in the lowest quartile of insulin sensitivity had lower concentrations of large and higher concentrations of small HDL than children in the top quartile (Figure 2A and B). Similarly, small dense LDL and very small LDL concentrations were higher in the lowest than the uppermost quartile of insulin sensitivity in both races (Figure 3A and B), irrespective of gender. Large VLDL and chylomicron concentration was significantly higher in the most insulin resistant quartile of black males \((P<0.05)\) and the bottom two quartiles of white children (both \(P<0.05)\) compared with the most insulin sensitive children in each group (Figure 3C).

After correcting for pubertal development across insulin sensitivity quartiles the significance for large HDL concentrations in black males changed from \(P=0.022\) to \(P=0.111\), and in black females the significance for small HDL changed from \(P=0.037\) to \(P=0.109\), and in white males it changed from \(P=0.027\) to \(P=0.078\). Excluding black or white girls with PCOS from their respective data sets did not change significance values across quartiles.

**Contribution of insulin sensitivity and Visceral Adiposity to lipoprotein particle size:** In multiple regression analyses with lipoprotein particle size as the dependent variable and race, gender, age, insulin sensitivity and VAT as the independent variables, VAT and insulin sensitivity independently and together explained 26% of the variance \((P< 0.001)\) in LDL size (VAT, partial \(r=-0.293, P<0.001\); insulin sensitivity, partial \(r=0.194, P=0.008\)) and 41% of the variance \((P< 0.001)\) in HDL size (VAT, partial \(r=-0.368, P<0.001\); insulin sensitivity, partial \(r=0.301, P<0.001\)), while VAT and race explained 12% of the variance \((P< 0.001)\)in VLDL size (VAT, partial \(r=-0.266, P<0.001\); race, partial \(r=0.199, P=0.007\)).

**CONCLUSIONS**

The present study advances previous observations of favorable lipoprotein profiles in black compared with white children \((3,6,8,14,15)\), and demonstrates that this is partly explained by lower visceral adiposity in blacks. Moreover, we show that in both racial groups the most insulin resistant youths have an atherogenic lipoprotein profile of small dense LDL, small HDL, and large VLDL, akin to the atherogenic pattern in adults with coronary artery disease \((10,11)\). Considering that atherosclerosis starts in childhood \((21)\),
such a lipoprotein pattern may have serious health consequences.

Our findings, from the whole group (Online Appendix Table A2), are consistent with the Bogalusa heart study (6,8) where black children had HDL and LDL particles on average 0.3 nm and 0.2 nm larger (6,8) and VLDL particles 3.6 nm smaller than their white peers (6). These values are similar to the mean differences we observed - 0.2 nm larger, 0.2nm larger and 3.8 nm smaller for HDL, LDL and VLDL in blacks, respectively. Several studies demonstrate favorable lipid profiles in black compared with white children (6,8,14,15) despite insulin resistance (12,13) with similar observations in adults (22). Importantly, for similar overall adiposity blacks have lower visceral adiposity than whites (3,15,20), an observation repeated in the current study. Controlling for VAT abolished black-white differences in LDL and HDL particle size and concentration in the present study. However, visceral adiposity did not account for the race-related differences in VLDL particle size and concentration which remained significant after adjusting for VAT. Two potential explanations for this could be a) the lower concentrations of TG in black children, since lipoprotein size is related to concentration (6); and b) increased lipoprotein TG clearance (22), as post heparin lipoprotein lipase activity is reported to be higher and hepatic lipase activity lower in black adults (22,23).

The present study suggests that gender-specific analyses, besides race, are important. Black males had smaller VLDL particles and black females had larger HDL particles than their white counterparts. For particle concentration race differences existed in the larger VLDL in both genders and for males only in the small dense LDL. One note of caution is that we report nominal significance values for race comparisons on lipid variables in both the group as a whole (Online Appendix Table 1A) and for gender-specific analyses (Table 2). The use of multiple t-tests may have increased the chance of finding a difference in our data. Nevertheless, significant race differences in some findings remain even if adjusted for multiple comparisons, particularly VLDL concentrations in both genders and LDL concentrations in males.

Our study is the first to examine the relationship between in vivo insulin sensitivity and lipoprotein particle size and subclass concentrations in children. The most insulin resistant children, irrespective of race or gender, had smaller LDL particles and higher concentrations of small dense LDL compared with their more insulin sensitive peers. A previous study found the prevalence of small dense LDL was 10% in children with insulin resistance syndrome (IRS) in contrast to 1% in those without IRS (7). However, IRS was defined based on fasting insulin. Small dense LDL particles are predictive of coronary heart disease (5,24). Conversely, large HDL particles have an inverse relationship with coronary heart disease whilst small HDL a positive association (25). Our data demonstrate that in both races insulin resistance was associated with small HDL particle size, increased small HDL concentration, and low large HDL concentration. Finally, VLDL particles differ in atherogenicity with some investigations suggesting large particles are most strongly related to arterial disease and obesity (25). Our data demonstrate that the more insulin resistant white and black children had higher concentrations of large VLDL and bigger VLDL particle size. Lastly, in multiple regression analyses both VAT and insulin sensitivity were significant determinants of LDL and HDL particle size, while VAT and race were for VLDL particle size.

In conclusion, our data confirm prior observations of favorable lipoprotein profiles in black youths compared with whites, and advance it by showing the role of the lower
visceral adiposity in blacks. Gender affects the extent of these differences. Moreover, in both blacks and whites the most insulin resistant youth exhibit small dense LDL, small HDL, and large VLDL profiles similar to the atherogenic lipoprotein pattern in adults with coronary artery disease (10,11). Such data underscore the need to initiate therapeutic interventions early in childhood to lessen abdominal obesity and insulin resistance, and improve the associated adverse alterations in lipoprotein profile, irrespective of race or gender, and reduce the potential risk of atherosclerotic cardiovascular changes.

ACKNOWLEDGEMENTS

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REFERENCES


Figure 1. HDL (panel A), LDL (panel B), and VLDL (panel C) particle size by quartiles of insulin sensitivity in black males (n=43; filled black bars) and females (n=57; dark black stripes), and white males (n=45; white bars) and females (n=49; light black stripes). Differences within each group compared using one-way ANOVA with post-hoc Tukey correction. a = Significant difference vs 4; b = Significant difference vs 3; c = Significant difference vs 2; P<0.05. (Range of insulin sensitivity for blacks: quartile 1: 0.45-1.95 ; 2 : 2.16-4.37 ; 3 : 4.40-9.20 ; 4 : 9.21-18.39 µmol/kg/min per pmol/L; and for whites quartile 1 : 0.68-1.75 ; 2 : 1.78-3.76 ; 3 : 3.80-9.21 ; 4 : 9.28-25.32 µmol/kg/min per pmol/L).

Figure 2. Concentrations of large (panel A), and small (panel B) HDL by quartiles of insulin sensitivity in black and white males and females. Differences compared using one-way ANOVA with post-hoc Tukey correction. a = Significant difference vs 4; b = Significant difference vs 3; c = Significant difference vs 2; P<0.05.

Figure 3. Concentrations of small (panel A), and very small (panel B) LDL by quartiles of insulin sensitivity in black and white males and females. Differences compared using one-way ANOVA with post-hoc Tukey correction. a = Significant difference vs 4; b = Significant difference vs 3; c = Significant difference vs 2; P<0.05.

Table 1. Physical characteristics of the participants

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
<th>Black vs White Males</th>
<th>Black vs White Females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blacks (n=49)</td>
<td>Whites (n=52)</td>
<td>Blacks (n=68)</td>
<td>Whites (n=57)</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>12.7 ± 0.3</td>
<td>13.5 ± 0.3</td>
<td>12.8 ± 0.3</td>
<td>12.6 ± 0.3</td>
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<tr>
<td>Tanner stage</td>
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<tr>
<td>I</td>
<td>9</td>
<td>6</td>
<td>10</td>
<td>8</td>
</tr>
<tr>
<td>II-III</td>
<td>22</td>
<td>26</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>IV-V</td>
<td>18</td>
<td>20</td>
<td>43</td>
<td>34</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>158.9 ± 2.0</td>
<td>164.5 ± 1.8</td>
<td>154.8 ± 1.3</td>
<td>154.8 ± 1.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.3 ± 3.9</td>
<td>76.1 ± 4.6</td>
<td>70.6 ± 3.4</td>
<td>69.5 ± 3.7</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.0 ± 1.1</td>
<td>27.1 ± 1.2</td>
<td>28.4 ± 1.1</td>
<td>28.2 ± 1.2</td>
</tr>
<tr>
<td>BMI Percentile</td>
<td>83.0 ± 3.1</td>
<td>79.3 ± 4.0</td>
<td>82.7 ± 3.0</td>
<td>84.2 ± 2.9</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>20.8 ± 2.2</td>
<td>21.5 ± 2.3</td>
<td>26.8 ± 2.0</td>
<td>25.7 ± 2.1</td>
</tr>
<tr>
<td>Fat free mass (kg)</td>
<td>43.9 ± 1.8</td>
<td>44.7 ± 1.9</td>
<td>40.0 ± 1.4</td>
<td>37.1 ± 1.3</td>
</tr>
<tr>
<td>Body fat %</td>
<td>27.9 ± 2.1</td>
<td>27.7 ± 1.8</td>
<td>35.3 ± 1.4</td>
<td>36.2 ± 1.5</td>
</tr>
<tr>
<td>Waist circ. (cm)</td>
<td>85.6 ± 3.0</td>
<td>90.8 ± 3.3</td>
<td>83.0 ± 2.9</td>
<td>80.1 ± 2.9</td>
</tr>
<tr>
<td>VAT (cm²)</td>
<td>39.4 ± 5.4</td>
<td>56.1 ± 6.7</td>
<td>37.3 ± 3.6</td>
<td>52.9 ± 5.4</td>
</tr>
<tr>
<td>SAT (cm²)</td>
<td>245.8 ± 32.3</td>
<td>303.9 ± 37.3</td>
<td>327.3 ± 29.0</td>
<td>339.2 ± 31.6</td>
</tr>
</tbody>
</table>

BMI, body mass index; VAT, visceral adipose tissue; SAT, subcutaneous adipose tissue. Tanner stages compared using Chi-square. All other variables compared using independent t-tests.
Table 2. Lipoprotein subclass concentration, particle size and plasma lipids in black versus white children.

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
<th>Black vs White Males</th>
<th>Black vs White Females</th>
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<tbody>
<tr>
<td></td>
<td>Blacks n=49</td>
<td>Whites n=52</td>
<td></td>
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<tr>
<td>Lipoprotein subclass concentration (nmol/L)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Total VLDL &amp; Chylomicrons</td>
<td>41.7 ± 2.9</td>
<td>54.8 ± 2.9</td>
<td>40.3 ± 2.2</td>
<td>53.5 ± 2.5</td>
</tr>
<tr>
<td>Large VLDL &amp; Chylomicrons</td>
<td>1.7 ± 0.4</td>
<td>4.1 ± 0.7</td>
<td>1.3 ± 0.3</td>
<td>2.7 ± 0.4</td>
</tr>
<tr>
<td>Medium VLDL</td>
<td>11.4 ± 1.5</td>
<td>19.4 ± 1.9</td>
<td>10.8 ± 1.1</td>
<td>18.3 ± 1.3</td>
</tr>
<tr>
<td>Small VLDL</td>
<td>28.6 ± 1.7</td>
<td>31.4 ± 2.1</td>
<td>28.2 ± 1.6</td>
<td>32.4 ± 1.6</td>
</tr>
<tr>
<td>LDL (nmol/L)</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Total LDL</td>
<td>833.0 ± 40.4</td>
<td>993.8 ± 53.3</td>
<td>828.6 ± 35.6</td>
<td>887.7 ± 43.9</td>
</tr>
<tr>
<td>Large LDL</td>
<td>316.1 ± 20.0</td>
<td>302.6 ± 22.2</td>
<td>321.3 ± 15.3</td>
<td>296.4 ± 16.4</td>
</tr>
<tr>
<td>Small LDL</td>
<td>481.6 ± 41.3</td>
<td>649.8 ± 55.3</td>
<td>474.5 ± 36.1</td>
<td>553.7 ± 48.3</td>
</tr>
<tr>
<td>Medium Small LDL</td>
<td>104.9 ± 8.4</td>
<td>133.8 ± 12.4</td>
<td>100.2 ± 7.1</td>
<td>116.4 ± 9.4</td>
</tr>
<tr>
<td>Very Small LDL</td>
<td>376.8 ± 33.2</td>
<td>515.9 ± 43.2</td>
<td>374.3 ± 29.3</td>
<td>437.3 ± 39.1</td>
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<tr>
<td>HDL (µmol/L)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Total HDL</td>
<td>27.1 ± 0.7</td>
<td>25.8 ± 0.7</td>
<td>25.1 ± 0.5</td>
<td>24.7 ± 0.5</td>
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<tr>
<td>Large HDL</td>
<td>6.2 ± 0.5</td>
<td>5.5 ± 0.5</td>
<td>6.2 ± 0.3</td>
<td>5.4 ± 0.4</td>
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<tr>
<td>Medium HDL</td>
<td>4.8 ± 0.5</td>
<td>4.5 ± 0.7</td>
<td>3.5 ± 0.4</td>
<td>3.7 ± 0.3</td>
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<tr>
<td>Small HDL</td>
<td>16.1 ± 0.6</td>
<td>15.8 ± 0.7</td>
<td>15.4 ± 0.6</td>
<td>15.6 ± 0.5</td>
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<tr>
<td>IDL (nmol/L)</td>
<td>35.3 ± 4.7</td>
<td>41.4 ± 7.4</td>
<td>32.7 ± 4.1</td>
<td>37.4 ± 4.8</td>
</tr>
<tr>
<td>Lipoprotein Particle Size (nm)</td>
<td></td>
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</tr>
<tr>
<td>VLDL</td>
<td>50.8 ± 1.2</td>
<td>56.4 ± 1.5</td>
<td>50.7 ± 1.3</td>
<td>52.8 ± 1.0</td>
</tr>
<tr>
<td>LDL</td>
<td>21.2 ± 0.1</td>
<td>20.9 ± 0.1</td>
<td>21.2 ± 0.1</td>
<td>21.1 ± 0.1</td>
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<tr>
<td>HDL</td>
<td>9.1 ± 0.1</td>
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<td>9.1 ± 0.1</td>
<td>8.9 ± 0.1</td>
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<td>Plasma Lipids (mmol/L)</td>
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<tr>
<td>Total Cholesterol</td>
<td>4.09 ± 0.12</td>
<td>4.49 ± 0.13</td>
<td>3.93 ± 0.09</td>
<td>4.12 ± 0.09</td>
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<tr>
<td>LDL Cholesterol</td>
<td>2.44 ± 0.10</td>
<td>2.66 ± 0.12</td>
<td>2.36 ± 0.09</td>
<td>2.45 ± 0.08</td>
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<tr>
<td>HDL Cholesterol</td>
<td>1.20 ± 0.05</td>
<td>1.16 ± 0.04</td>
<td>1.18 ± 0.03</td>
<td>1.11 ± 0.03</td>
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<tr>
<td>Total TG</td>
<td>1.00 ± 0.08</td>
<td>1.50 ± 0.14</td>
<td>0.94 ± 0.07</td>
<td>1.26 ± 0.08</td>
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<tr>
<td>VLDL TG</td>
<td>0.20 ± 0.02</td>
<td>0.30 ± 0.03</td>
<td>0.19 ± 0.01</td>
<td>0.25 ± 0.02</td>
</tr>
</tbody>
</table>

VLDL, very-low-density lipoprotein; LDL, low-density-lipoprotein; IDL, intermediate density lipoprotein; HDL, high density lipoprotein; TG, triglyceride.

All comparisons made using independent t-tests. Nominal p values indicate that significance values are unadjusted for multiple comparisons.
Figure 1

A) Black
Males P=0.004
Females P<0.001
All P<0.001

B) Males P=0.010
Females P<0.001
All P<0.001

C) Males P=ns
Females P=ns
All P=0.022

VLDL Particle Size (nm)

Quartiles of Insulin Sensitivity

HDL Particle Size (nm)

LDL Particle Size (nm)

Black White

Males P=0.004
Females P<0.001
All P<0.001

Males P=0.010
Females P<0.001
All P<0.001

Males P=ns
Females P=ns
All P=0.022

Males P<0.001
Females P=0.008
All P<0.001

Males P=0.001
Females P=ns
All=0.004
Figure 2

**Black**
- Males $P=0.022$
- Females $P<0.001$
- All $P<0.001$

**White**
- Males $P=0.004$
- Females $P=0.393$
- All $P=0.001$

Figure 3

**Black**
- Males $P=0.003$
- Females $P<0.001$
- All $P<0.001$

**White**
- Males $P<0.001$
- Females $P=0.002$
- All $P<0.001$